09/419,901
updated Search
LYCook 6/16/05

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(FILE 'HOME' ENTERED AT 16:21:05 ON 16 JUN 2005)

FILE 'STNGUIDE' ENTERED AT 16:21:13 ON 16 JUN 2005

FILE 'HOME' ENTERED AT 16:21:22 ON 16 JUN 2005

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT 16:21:43 ON 16 JUN 2005

L1	304	S	(MYOFILAMENT PROTEIN)
L2	159	S	(CHEMICAL ADDUCT)
L3	1	S	L1 AND L2
L4	180	S	L1 AND TROPONIN?
L5	95	S	L1 AND ACTIN?
L6	12	S	L1 AND DESMIN?
L7	110	S	L1 AND MYOSIN?
L8	1	S	L4 AND ADDUCT?
L9	1	S	L5 AND ADDUCT?
L10	1	S	L6 AND ADDUCT?
L11	101	S	L4 AND MUSCLE?
L12	1	S	L2 AND TROPONIN?
L13	15	S	L2 AND REVIEW?
L14	10	DUPLICATE REMOVE	L13 (5 DUPLICATES REMOVED)
L15	14	S	L2 AND ACTIN?
L16	1	S	L2 AND MYOSIN?
L17	1	S	L2 AND DESMIN?
L18	5	DUPLICATE REMOVE	L15 (9 DUPLICATES REMOVED)
L19	62	S	L5 AND MUSCLE?
L20	30	DUPLICATE REMOVE	L19 (32 DUPLICATES REMOVED)
L21	0	S	L20 AND TRANSLATION?
L22	11	S	L1 AND TRANSLATION?
L23	0	S	L22 AND ADDUCT?
L24	1	S	L1 AND ADDUCT?
L25	7	DUPLICATE REMOVE	L22 (4 DUPLICATES REMOVED)

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L24            1 S L1 AND ADDUCT?
L25            7 DUPLICATE REMOVE L22 (4 DUPLICATES REMOVED)

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ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
AN 1994:187066 BIOSIS
DN PREV199497200066
TI Detection of reduced acetaldehyde protein adducts using a unique monoclonal antibody.
AU Klassen, Lynell W. [Reprint author]; Tuma, Dean J.; Sorrell, Michael F.; McDonald, Thomas L.; Devasure, Jane M.; Thiele, Geoffrey M.
CS University Nebraska Medical Center, Dep. Internal Medicine, 600 S. 42nd St., Omaha, NE 68198-3332, USA
SO Alcoholism Clinical and Experimental Research, (1994) Vol. 18, No. 1, pp. 164-171.
CODEN: ACRSDM. ISSN: 0145-6008.
DT Article
LA English
ED Entered STN: 26 Apr 1994
Last Updated on STN: 27 Apr 1994
AB Acetaldehyde (AA), the major product of alcohol metabolism, has been shown to bind to proteins In vivo and form **chemical adducts**. These AA-protein adducts have been shown to alter protein structure and function and may result in tissue damage. Recent reports have shown that polyclonal antibodies can be produced that recognize proteins modified in vitro with AA in the presence of sodium cyanoborohydride (NaCNBH-3), a strong reducing (R) agent. Antibodies prepared in this way have been shown to recognize proteins in the livers of rats fed alcohol chronically. Because multiple AA-protein adducts can be recognized by polyclonal antisera, and a variety of adducts may be formed in vitro or in vivo, this study was designed to develop monoclonal antibodies specific for proteins modified by AA. In addition, adducts formed under R conditions are probably chemically different than those formed under nonreducing (NR) conditions, and monoclonal antibodies may provide the specificity required to distinguish these chemical differences. Balb/c mice were immunized with bovine brain tubulin that was modified by treatment with 5 mM AA for 7 days under NR conditions. Sera from immunized animals were tested for antibody activity to the immunogen (protein-NR) and for cross-reactivity to protein-R and unmodified protein. Although the highest serum antibody titers were seen toward the NR adduct, antibodies to the R adduct were also detected. This activity difference was independent of the carrier protein, because NR and R bovine serum albumin, keyhole limpet hemocyanin, and **actin** also gave similar results when used as the adducted protein. Surprisingly, all the monoclonal antibodies (RT1.1, RT1.2, RT1.3, and RT1.4) produced by hybridomas generated from spleen cells from NR-tubulin immunized mice recognized the R and not the NR adduct. One of these hybridomas (RT1.1) produces an IgG2b antibody that reacts with all tested proteins that have been modified with AA under chemical R conditions. Because of its monoclonal specificity, this antibody may be useful in probing for the presence of R AA-protein adducts made both in vitro and in vivo.
CC Biochemistry methods - General 10050
Biochemistry studies - General 10060
Enzymes - Methods 10804
Metabolism - General metabolism and metabolic pathways 13002
Toxicology - General and methods 22501
Immunology - General and methods 34502
IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Immune System (Chemical Coordination and Homeostasis); Metabolism; Toxicology
IT Chemicals & Biochemicals
ACETALDEHYDE; ALCOHOL
IT Miscellaneous Descriptors
ALCOHOL; ANALYTICAL METHOD; ELISA; TOXICOKINETICS
ORGN Classifier

Bovidae 85715
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
bovine
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
ORGN Classifier
Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
rabbit
Taxa Notes
Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 75-07-0 (ACETALDEHYDE)
64-17-5 (ALCOHOL)

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Metabolism - General metabolism and metabolic pathways 13002
Toxicology - General and methods 22501
Immunology - General and methods 34502
IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and Molecular Biophysics); Immune System (Chemical Coordination and Homeostasis); Metabolism; Toxicology
IT Chemicals & Biochemicals
ACETALDEHYDE; ALCOHOL
IT Miscellaneous Descriptors
ALCOHOL; ANALYTICAL METHOD; ELISA; TOXICOKINETICS
ORGN Classifier

Bovidae 85715
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
bovine
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
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Leporidae 86040
Super Taxa
Lagomorpha; Mammalia; Vertebrata; Chordata; Animalia
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Animals, Chordates, Lagomorphs, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Vertebrates
RN 75-07-0 (ACETALDEHYDE)
64-17-5 (ALCOHOL)

ANSWER 28 OF 30 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 86145956 EMBASE
DN 1986145956
TI Thin **myofilament proteins** in norm and heart failure.
I. Polymerizability of myocardial Straub **actin** in acute and chronic heart failure.
AU Karsanov N.V.; Pirtskhalaishvili M.P.; Semerikova V.J.; Losaberidze Sh. N.
CS The Republican Centre of Medical Biophysics of the Georgian SSR Public Health Ministry, 380092 Tbilisi, Russia
SO Basic Research in Cardiology, (1986) Vol. 81, No. 2, pp. 199-212.
CODEN: BRCAB7
CY Germany
DT Journal
FS 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LA English
ED Entered STN: 911210
Last Updated on STN: 911210
AB The reduced and intrinsic viscosities of myocardial Straub F-**actin** from the left ventricle of a practically healthy man were equal to 3.05±0.2 and 2.4±0.32 and from the right ventricle were 2.37±0.2 and 2.1±0.3 dl/g, respectively (the difference between ventricles was not significant). The average length of filaments measured by flow birefringence technique was equal to 1.3±0.04 μm, the number-average length (<L>n), determined by the electron microscopy was 1.4 μm, the weight-average length (<L>w), was 2 μm and the maximal one was 5.5 μm. The histograms showed that the most characteristic length was that of 0.8-1.2 μm. According to the flow birefringence data canine myocardial F-**actin** had a length similar to that of myocardial F-**actin** from a practically healthy man, though its reduced and intrinsic viscosities were higher. In acute and especially chronic congestive heart failure the **actin** polymerizability was sharply reduced. In consequence, in acute heart failure the number-average length of F-**actin** filaments was decreased by 43% and in congestive heart failure by 65.7%. The characteristic length in acute heart failure shifts to the range of 0.2-0.6 μm, while in congestive heart failure the range is 0.2-0.4 μm. This fact can possibly explain why during preparation of **actin** from the pathologically changed myocardium according to the methods including purification by the cycles of polymerization-sedimentation-depolymerization, the pathologically changed **actin** is discarded and the normal **actin** remains. A definite parallel was observed between the reduction of **actin** polymerizability and the ability of myocardial glucerinated fiber bundles (MBGF) to generate force. We conclude that the changes of **actin** properties in heart failure may cause a decrease in contractibility of the myocardial contractile protein system.
CT Medical Descriptors:
*heart failure
***heart muscle cell**
*myofilament
dog
electron microscopy
human
priority journal
etiology
diagnosis
clinical article
animal experiment
nonhuman
heart
muscle
Drug Descriptors:

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nonhuman
heart
muscle
Drug Descriptors:

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*myofilament
dog
electron microscopy
human
priority journal
etiology
diagnosis
clinical article
animal experiment
nonhuman
heart
muscle
Drug Descriptors:

ANSWER 25 OF 30 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 12

AN 1993:101018 BIOSIS

DN PREV199395056214

TI Estrogen-induced morphological and immunohistochemical changes in stroma and epithelium of rat ventral prostate.

AU Zhao, G. W.; Holterhus, P. M.; Dammshaeuser, I.; Hoffbauer, G.; Aumueller, G. [Reprint author]

CS Dep. Anat. Cell Biol., Robert-Koch-Str., D-3550 Marburg, Germany

SO Prostate, (1992) Vol. 21, No. 3, pp. 183-199.
CODEN: PRSTDS. ISSN: 0270-4137.

DT Article

LA English

ED Entered STN: 9 Feb 1993
Last Updated on STN: 10 Feb 1993

AB Prostatic smooth **muscle** cells have been regarded to play a major pathogenetic role during the development of benign prostatic hyperplasia (BPH) in elderly men. Altered hormonal signals (increased estrogen) have been made responsible for the "metabolic" transformation of prostatic smooth **muscle** cells, which were thought to produce increased amounts of connective tissue fibers observed in BPH. In order to find out the role of metabolically "activated" smooth **muscle** cells, hormone stimulation experiments were performed in male rats. The effects of androgen deprivation and estrogen stimulation were recorded by semiquantitative analysis of intermediate and **myofilament proteins** in stromal smooth **muscle** cells. In castrated or estrogen-treated or estrogen-treated and castrated animals, the reduction of the glandular lumen is the most obvious morphological alteration, accompanied by an increase in connective tissue. Regressive changes occurred most rapidly in castrated animals (already within the first week), slower in castrated estrogen-treated animals and still slower in normal estrogen-treated animals. Regression of the epithelium was accompanied by a marked decrease in immunoreactivity for prostatic binding protein (PBP) in castrated animals, while PBP immunoreactivity in estrogenized animals was retained for up to 6 weeks. Smooth **muscle** cells became atrophic in castrated animals. This effect was attenuated in estrogen-treated animals. There was no indication for enhanced collagen synthesis by smooth **muscle** cells. **Actin** and desmin-immunoreactivity were only slightly altered in experimental animals and showed a changed distribution pattern. Prostatic smooth **muscle** cells respond less markedly to hormonal alterations than do the fibroblasts.

CC Microscopy - Histology and histochemistry 01056
Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Sterols and steroids 10067
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Anatomy 15502
Urinary system - Physiology and biochemistry 15504
Urinary system - Pathology 15506
Reproductive system - Anatomy 16502
Reproductive system - Physiology and biochemistry 16504
Reproductive system - Pathology 16506
Endocrine - Gonads and placenta 17006
Muscle - Physiology and biochemistry 17504
Gerontology - 24500

IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Muscular System (Movement and Support); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis); Urology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals
ACTIN
IT Miscellaneous Descriptors
ACTIN; BENIGN PROSTATIC HYPERPLASIA; COLLAGEN; DESMIN; ELDERLY; PROSTATIC BINDING PROTEIN; PROSTATIC SMOOTH **MUSCLE** CELLS
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 132579-20-5 (**ACTIN**)

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AN 1993:101018 BIOSIS

DN PREV199395056214

TI Estrogen-induced morphological and immunohistochemical changes in stroma and epithelium of rat ventral prostate.

AU Zhao, G. W.; Holterhus, P. M.; Dammshaeuser, I.; Hoffbauer, G.; Aumueller, G. [Reprint author]

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Reproductive system - Anatomy 16502
Reproductive system - Physiology and biochemistry 16504
Reproductive system - Pathology 16506
Endocrine - Gonads and placenta 17006
Muscle - Physiology and biochemistry 17504
Gerontology - 24500

IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Muscular System (Movement and Support); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis); Urology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals
ACTIN
IT Miscellaneous Descriptors
ACTIN; BENIGN PROSTATIC HYPERPLASIA; COLLAGEN; DESMIN; ELDERLY; PROSTATIC BINDING PROTEIN; PROSTATIC SMOOTH **MUSCLE** CELLS
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 132579-20-5 (**ACTIN**)

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AN 1993:101018 BIOSIS

DN PREV199395056214

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CS Dep. Anat. Cell Biol., Robert-Koch-Str., D-3550 Marburg, Germany

SO Prostate, (1992) Vol. 21, No. 3, pp. 183-199.
CODEN: PRSTDS. ISSN: 0270-4137.

DT Article

LA English

ED Entered STN: 9 Feb 1993
Last Updated on STN: 10 Feb 1993

AB Prostatic smooth **muscle** cells have been regarded to play a major pathogenetic role during the development of benign prostatic hyperplasia (BPH) in elderly men. Altered hormonal signals (increased estrogen) have been made responsible for the "metabolic" transformation of prostatic smooth **muscle** cells, which were thought to produce increased amounts of connective tissue fibers observed in BPH. In order to find out the role of metabolically "activated" smooth **muscle** cells, hormone stimulation experiments were performed in male rats. The effects of androgen deprivation and estrogen stimulation were recorded by semiquantitative analysis of intermediate and **myofilament proteins** in stromal smooth **muscle** cells. In castrated or estrogen-treated or estrogen-treated and castrated animals, the reduction of the glandular lumen is the most obvious morphological alteration, accompanied by an increase in connective tissue. Regressive changes occurred most rapidly in castrated animals (already within the first week), slower in castrated estrogen-treated animals and still slower in normal estrogen-treated animals. Regression of the epithelium was accompanied by a marked decrease in immunoreactivity for prostatic binding protein (PBP) in castrated animals, while PBP immunoreactivity in estrogenized animals was retained for up to 6 weeks. Smooth **muscle** cells became atrophic in castrated animals. This effect was attenuated in estrogen-treated animals. There was no indication for enhanced collagen synthesis by smooth **muscle** cells. **Actin** and desmin-immunoreactivity were only slightly altered in experimental animals and showed a changed distribution pattern. Prostatic smooth **muscle** cells respond less markedly to hormonal alterations than do the fibroblasts.

CC Microscopy - Histology and histochemistry 01056
Cytology - Animal 02506
Cytology - Human 02508
Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Sterols and steroids 10067
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Anatomy 15502
Urinary system - Physiology and biochemistry 15504
Urinary system - Pathology 15506
Reproductive system - Anatomy 16502
Reproductive system - Physiology and biochemistry 16504
Reproductive system - Pathology 16506
Endocrine - Gonads and placenta 17006
Muscle - Physiology and biochemistry 17504
Gerontology - 24500

IT Major Concepts
Endocrine System (Chemical Coordination and Homeostasis); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Muscular System (Movement and Support); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis); Urology (Human Medicine, Medical Sciences)

IT Chemicals & Biochemicals
ACTIN
IT Miscellaneous Descriptors
ACTIN; BENIGN PROSTATIC HYPERPLASIA; COLLAGEN; DESMIN; ELDERLY; PROSTATIC BINDING PROTEIN; PROSTATIC SMOOTH **MUSCLE** CELLS
ORGN Classifier
Hominidae 86215
Super Taxa
Primates; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
human
Taxa Notes
Animals, Chordates, Humans, Mammals, Primates, Vertebrates
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Muridae
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates
RN 132579-20-5 (**ACTIN**)

ANSWER 21 OF 30 EMBASE
on STN
AN 95289839 EMBASE
DN 1995289839
TI Troponin C - Troponin I interactions and molecular signalling in cardiac myofilaments.
AU Solaro R.J.; Lab M.; Landesberg A.; Burkhoff D.; Ter Keurs H.
CS Department of Physiology/Biophysics, College of Medicine, University of Illinois, 901 South Wolcott,Chicago, IL 60612-7342, United States
SO Advances in Experimental Medicine and Biology, (1995) Vol. 382, pp. 109-115.
ISSN: 0065-2598 CODEN: AEMBAP
CY United States
DT Journal; Conference Article
FS 002 Physiology
018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LA English
SL English
ED Entered STN: 951017
Last Updated on STN: 951017
AB This chapter describes a current perception of the molecular interactions regulating myofilament activity in heart cells. The focus is on the interaction between troponin-C (TnC), the Ca2+-receptor and troponin I (TnI), an inhibitory protein. It is this interaction that appears to form a molecular switch that turns on the thin filament. It will be seen that control of the **actin**-myosin reaction is not only through Ca2+-binding to TnC, but also through steric, cooperative and allosteric processes involving all of the main **myofilament proteins** -**actin**, myosin, tropomyosin (Tm), troponin T (TnT), TnC, and TnI. The process is modulated by covalent and non- covalent mechanisms. The process is altered in diverse myopathies and pathologies of the heart and is a target for pharmacological manipulation by a new class of inotropic agents, the 'Ca2+-sensitizers'.
CT Medical Descriptors:
***actin myosin interaction**
*cardiomyopathy: DT, drug therapy
*cardiomyopathy: ET, etiology
*molecular interaction
allosterism
calcium binding
conference paper
heart muscle cell
myofilament
priority journal
protein phosphorylation
signal transduction
Drug Descriptors:
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*inotropic agent: DT, drug therapy
*inotropic agent: PD, pharmacology
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*troponin i
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myosin
protein kinase c
tropomyosin
troponin t
RN (calcium ion) 14127-61-8; (troponin c) 56094-11-2; (troponin i) 77108-40-8; (protein kinase c) 141436-78-4; (tropomyosin) 72067-79-9; (troponin t) 60304-72-5

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on STN
AN 1998056030 EMBASE
TI Breakdown and release of **myofilament proteins** during ischemia and ischemia/reperfusion in rat hearts identification of degradation products and effects on the pCa-force relation.
AU Van Eyk J.E.; Powers F.; Law W.; Larue C.; Hodges R.S.; Solaro R.J.
CS Dr. J.E. Van Eyk, Department of Physiology, Queen's University, Kingston, Ont. K7L 3W6, Canada
SO Circulation Research, (9 Feb 1998) Vol. 82, No. 2, pp. 261-271.
Refs: 56
ISSN: 0009-7330 CODEN: CIRUAL
CY United States
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
LA English
SL English
ED Entered STN: 19980312
Last Updated on STN: 19980312
AB Our objective in experiments reported here was to identify **myofilament proteins** of rat hearts either lost or degraded by cardiac ischemia (15- or 60-minute duration) with and without 45 minutes of reperfusion. We correlated these changes with alterations in myofilament sensitivity to Ca2+ and maximum force generation. Protein degradation and loss were assessed by high- performance liquid chromatography, SDS-PAGE, Western blotting analysis, and amino acid sequencing. Compared with nonischemic control hearts, bundles of skinned fibers from hearts subjected to ischemia alone demonstrated a decrease in maximum force generation and an increase in sensitivity to Ca2+. These changes in function were increased with the duration of the ischemia and with reperfusion. With increasing duration of ischemia, there was an increased loss and degradation of myofibrillar α- **actinin** and troponin I (TnI) at its C-terminus. α- **Actinin** and TnI were most susceptible to ischemia, but with 60 minutes of ischemia/reperfusion, there was also degradation of myosin light chain-1 (MLC1) involving a clip of residues 1 to 19. The MLC1 degradation product was detected in the reperfusion effluent (along with troponin T, tropomyosin, and α- **actinin**) but not in the tissue with 60 minutes of ischemia with no reperfusion. Moreover, with ischemia the following proteins became associated with the myofibrils: GAPDH and proteins of the mitochondrial ATP synthase complex. Our results provide new evidence regarding the mechanism by which ischemia/reperfusion causes myocardial injury and support the hypothesis that an important element in the injury is altered activity and structure of the myofilaments.
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***heart muscle reperfusion**
*reperfusion injury: ET, etiology
pathophysiology
protein secretion
myofilament
protein degradation
calcium cell level
nonhuman
rat
animal tissue
article
priority journal
Drug Descriptors:
troponin i: EC, endogenous compound
tropomyosin: EC, endogenous compound
alpha actin: EC, endogenous compound

RN (troponin i) 77108-40-8; (tropomyosin) 72067-79-9

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